Fig. 1

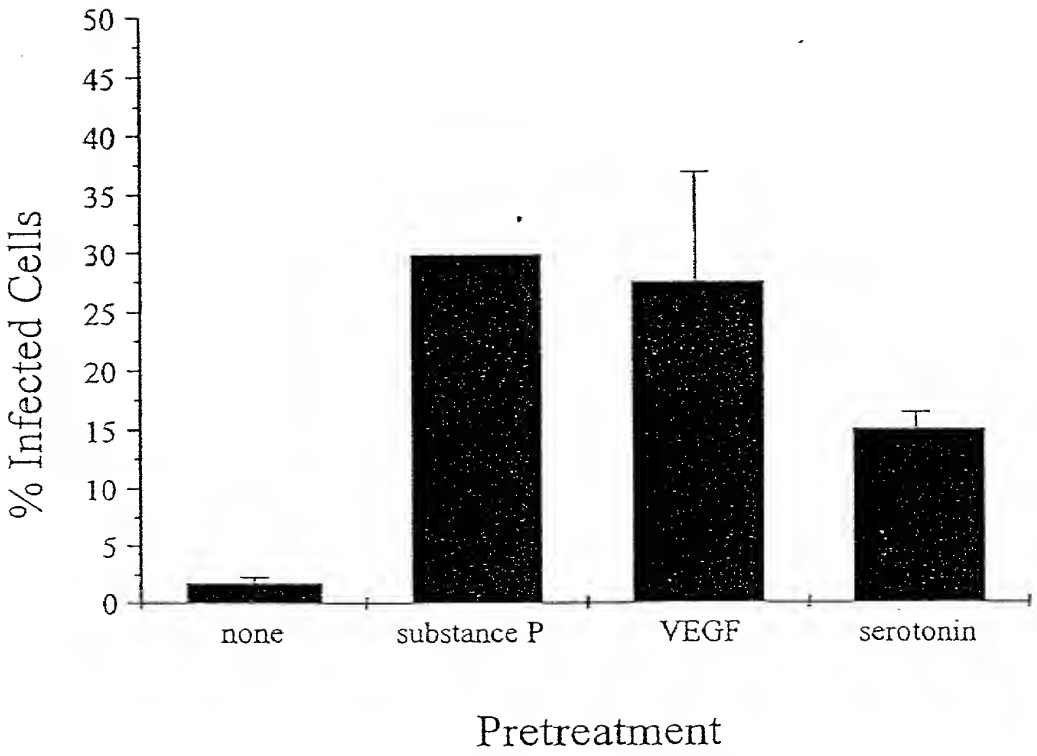
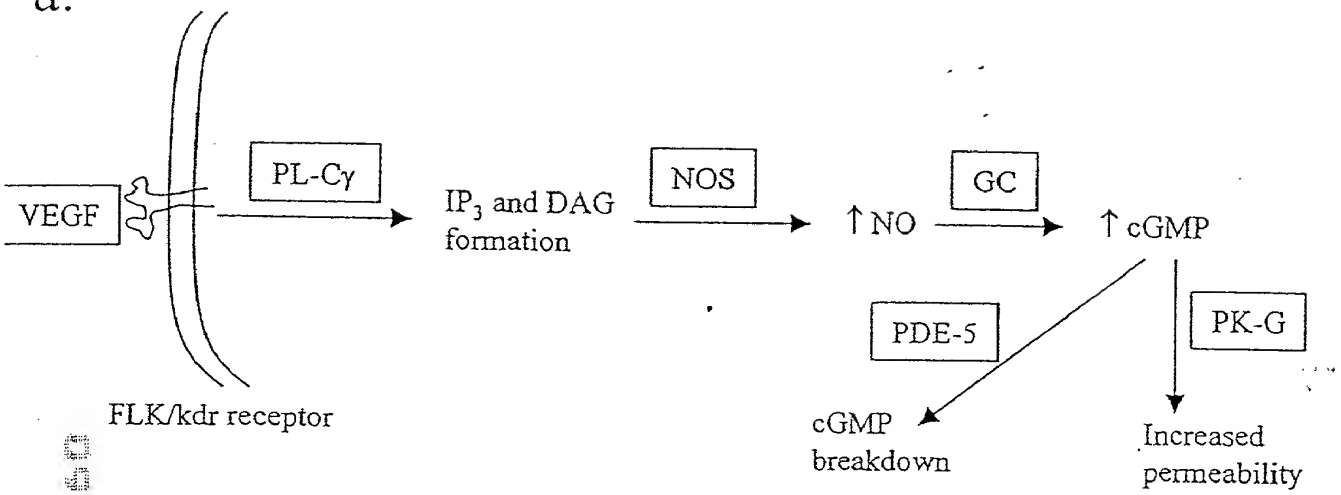


Fig. 1. Effect of pretreatment on adenoviral gene transfer. *Ex vivo* perfused hearts were exposed to substance P (1×10^{-7} M, 30 sec), VEGF (1×10^{-9} M, 2 min) or serotonin (1×10^{-5} M, 15 min) before 2 min Adβgal infection (1×10^{8} pfu/ml). n = 3 for each, except n = 1 for substance P.

Fig. 2

a.



b.

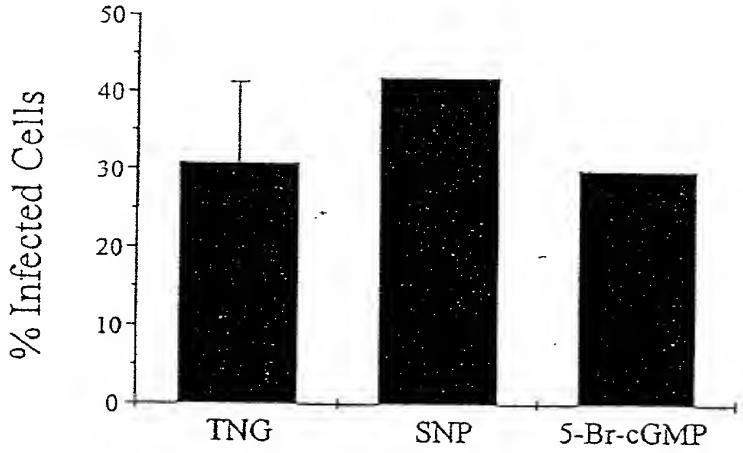


Fig. 2. Investigation of intracellular pathways mediating increase in vascular permeability and gene transfer. **a.** Schematic of intracellular pathway responsible for increases in vascular permeability. **b.** Effect of perfusion with nitroglycerin (TNG) or nitroprusside (SNP) or with 5-Br-cGMP on adenovirus-mediated gene transfer. TNG and SNP increase intracellular NO, and 5-Br-cGMP increases intracellular cGMP. n = 4 for TNG and n = 1 for SNP and 5-Br-cGMP Abbreviations: PL-Cγ: phospholipase C-γ, NOS: nitric oxide synthase, PDE-5: phosphodiesterase 5, GC: guanylate cyclase, PK-G: protein kinase G

Fig. 3

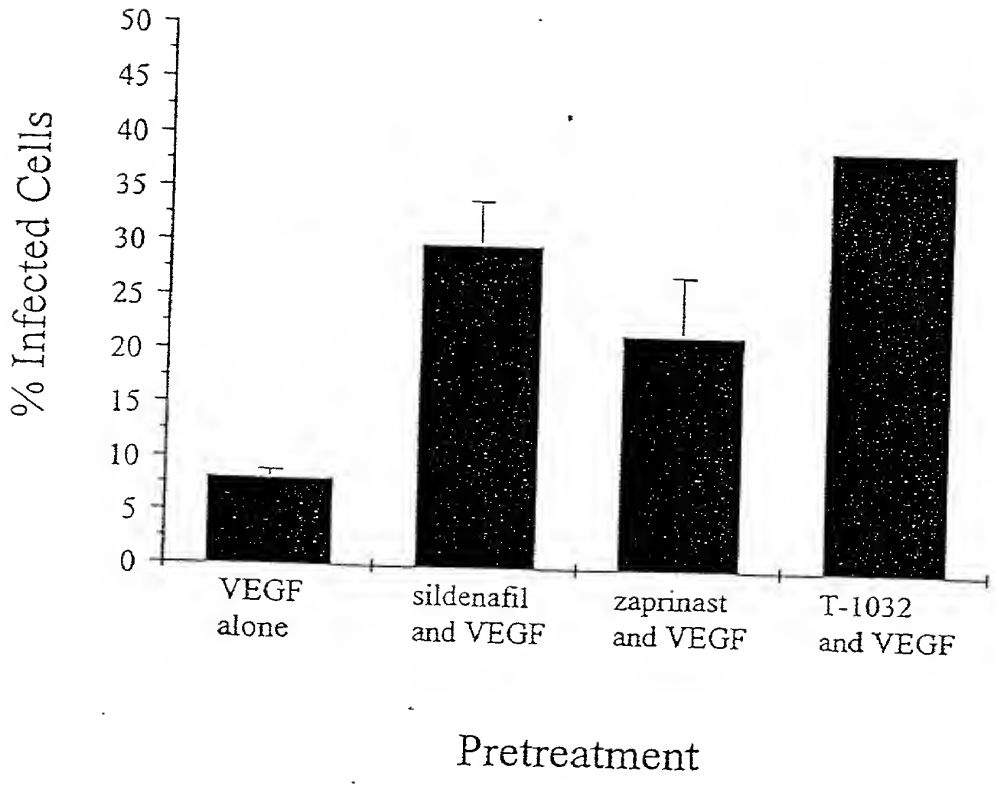


Fig. 3. Effect of phosphodiesterase 5 inhibition on adenoviral gene transfer. *Ex vivo* perfused hearts are exposed to VEGF (0.3×10^{10} M, 2 min) alone or after 15 min exposure to the PDE-5 inhibitors sildenafil (1×10^{-5} M), zaprinast (1×10^{-5} M) or T-1032 (1×10^{-6} M). Hearts were then exposed to Adβgal (1×10^{8} pfu/ml, 2 min), and the percentage of cells receiving the transgene was quantified. n = 3 for each except n = 1 for T-1032.